PB81-235772

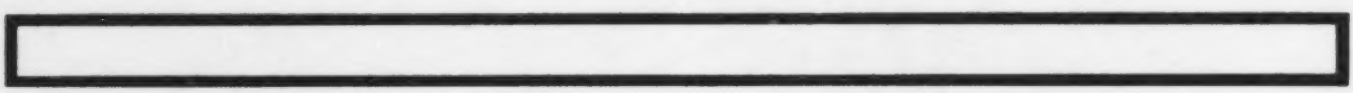
Drinking Water Sodium and Blood Pressure in Children: A Second Look

Massachusetts Univ., Amherst

Prepared for

Health Effects Research Lab.
Cincinnati, OH

Jul 81



U.S. Department of Commerce
National Technical Information Service
NTIS

EPA-600/J-81-289
Journal Article

Drinking Water Sodium and Blood Pressure in Children: A Second Look

ROBERT W. TUTHILL, PHD, AND EDWARD J. CALABRESE, PHD

Abstract: A previous study by the current authors demonstrated a statistically significant and clinically important elevation of 3–5 mmHg in mean systolic and diastolic blood pressure in high school sophomores in a community with 108 mg/L of sodium in the water supply when compared to their peers in an appropriately matched community with 8 mg/L of sodium. The current investigation, employing identical techniques but studying third graders in the same two communities, showed similar results. This second look considered dietary intake and urinary excretion of sodium. Since the difference in 24-hour dietary sodium consumption was 300 milligrams between the communities, an intake of one liter of high sodium tap water represented approximately 25 per cent of the difference in total sodium intake between the two communities. These studies suggest that sodium consumption in both drinking water and diet may be contributing to the difference in blood pressure distributions among the normotensive children in the two communities. (*Am J Public Health* 1981;71:722–729.)

Introduction

Sodium (Na) consumed in excess of physiological need has long been considered important in inducing an age-related increase in blood pressure (BP) that culminates in hypertension for the more genetically susceptible individuals. Animal research using rat, chicken, rabbit, dog, and monkey models has clearly demonstrated the hypertensinogenic effect of excess salt intake[1–17] The isolation of strains of rats predisposed to salt-induced hypertension has verified that a genetic factor is operative in the response to excess salt intake.[13–16] Primitive societies do not show an increase in blood pressure with age and consequently do not suffer the problem of hypertension[18–24] A factor common to all of these primitive groups is a low intake of salt. Among western nations, the higher the salt intake in the diet the higher the hypertension rate.[25, 26]

In human beings, severe sodium restriction has been shown to alleviate the symptoms of hypertension; upon reintroduction of sodium to the diet, blood pressure return to elevated levels.[27–31] The physiological mechanisms for the elevations in blood pressure are not completely understood although hemodynamic studies suggest that the development of hypertension may be a homeostatic response to sodium-induced expansion of the extracellular fluid volume.[32, 33]

The average daily human dietary intake of sodium in the United States of 2.8 to 6.0 grams is greatly in excess of the physiological requirements estimated to be 0.4 to 0.8 gm/day Na.[26, 34] Since drinking water contributes only from 0.5 to 9.0 per cent of the usual daily sodium intake of human beings (with the possible exception of those persons on a low salt diet),[35] it is understandable that this route of exposure remains largely unexplored. Therefore, the U.S. Environmental Protection Agency did not institute a maximum contaminant level for sodium in the National Safe Water Drinking Act of 1974.[36] However, the American Heart Association has implied that a limit of 20 mg/L Na is desirable as a standard to protect those suffering from heart of kidney ailments requiring a low sodium diet.[37]

Wolfe and Esmond reported a concomitant variation between sodium concentration in public drinking water in Dallas and arteriosclerotic and hypertensive heart disease.[38]

From the Division of Public Health, School of Health Sciences, University of Massachusetts. Address reprint requests to Robert W. Tuthill, PhD, Associate Professor, Division of Public Health, School of Health Sciences, University of Massachusetts, Amherst, MA 01003. This paper, submitted to the Journal May 12, 1980, was revised and accepted for publication December 5, 1980.

Editors' Note: See also related article, p. 729 this issue.

Similarly, studies from Romania and the Soviet Union have reported an increase prevalence of hypertension in communities with very high levels of sodium in the drinking water.[39, 40] However, the methodology and the data were incompletely reported in the Balkan studies, creating difficulty in interpreting the results.

A recent study by the current authors[41] demonstrated a statistically significant and clinically important upshift of 3–5 mmHg in mean blood pressures between high school sophomores in a community with 108 mg/L of sodium in the water supply when compared to their peers in an appropriately matched community with 8 mg/L. The high sodium community females exhibited a blood pressure pattern characteristic of persons ten years older, while for the males the upshift was similar to that expected for persons approximately two years older.

The current study is a repetition of the earlier investigation following identical techniques but studying third graders in the same two communities. The potential of illicit drug use was minimal in this age group and blood pressure was therefore, not likely to be differentially affected. In addition, urinary output of sodium and potassium and dietary intake of the same two elements were included in the study to document any differences in these cation levels between the communities. During this second year the average sodium level in the high sodium community was 120 mg/L because of the closing down of one well field.

Methodology

Blood Pressure

A variety of factors are known to affect the reliability and validity of blood pressure measurements. For this reason, the measurements were carried out in a highly standardized fashion and special care was taken to see that conditions were uniform among schools. A detailed description of the blood pressure procedures is presented in an earlier publication.[41] However, it is especially important to note that the same nurses took the blood pressure in both communities, each being scheduled for the same amount of time in each community. The screening of the seven schools from each community was planned in such a way that nurses kept going back and forth between the towns within the same day. This pattern was designed to allow the same proportion of pupils to be screened in the morning and afternoon in both systems to control for diurnal variation in blood pressure. Three successive casual seated blood pressures were recorded on every pupil by separate nurses. The three nurses were blind to each other's readings.

Selection of Communities

The two geographically contiguous communities were originally chosen because of a marked similarity in sociodemographic characteristics known to be associated with BP. The communities were similar in size (both in low 20,000s), median income, education, and ethnic composition, as determined by 1970 United States Census data. Of prime importance for experimental purposes was a difference of drinking water sodium levels between the two communities of 100 mg/L.

Control Variables

Information on other factors related to blood pressure was obtained for each child from a short questionnaire completed by the parents in conjunction with the permission slip. The questionnaire provided information on family history of high blood pressure, length of residence in town, medications being taken by the child, feeding habits as an infant, and the educational level and occupation of the main wage earner in the family.

Height and weight data were available from school health records compiled in the two months immediately preceding this study. The school scales were checked for consistency and all weighed accurately to within one pound of a 40-pound weight.

Dietary Diary

In addition, each child was asked to fill out a 24-hour dietary diary which was begun in school after lunch with the teacher's help and completed at home with the parent's aid. The record indicated the portion size, whether the food was fresh, frozen, or canned, the quantity of salt added in cooking, and the amount and type of liquid consumed. The diaries were obtained several weeks after the blood pressures were recorded.

The individual dietary items reported by each student were coded for nutrient content using the US Department of Agriculture (USDA) Handbook No. 456, "Nutritive Value of American Foods." For items not included in this source, a supplemental data bank was consulted. When no value could be found for a particular consumed item, that item was excluded from the calculations. This method relies on standard values rather than laboratory analyses of actual food consumed, thus only estimating an individual's intake of nutriments.

Urine Samples

A sub-set of 100 individuals from each community was asked for a first morning clean-catch urine sample. The 50 boys and 50 girls were selected from each community from a grid where systolic and diastolic blood pressure were cross-classified simultaneously by quintiles. Each cell of the grid was sampled in the same proportion as it was represented in the total group. The specimens were picked up from the doorstep of the children's homes by 8:00 am and were immediately refrigerated. All specimens were subsequently analyzed by flame photometry for sodium and potassium concentration within ten hours of collection. It was not feasible to obtain the urine samples on the same day as the dietary diary or the blood pressure determination.

Results

Blood Pressure

As a result of the excellent cooperation of school personnel at all levels, and the favorable response of the par-

ents, the final blood pressure screening rates of the third graders in the high and low sodium communities were 90.1 and 87.0 per cent, respectively.

The mean of the three blood pressure readings for each child was used for all analyses. Figure 1 shows a clear upshift in the blood pressure distributions of the high sodium community pupils when compared to their peers from the low sodium community. The upshift was consistent for systolic and diastolic blood pressures for both sexes.

Table 1 Section A indicates more specifically the mean differences in blood pressures between the children of the two communities. For boys, the systolic mean difference was 3.2 mmHg with a p of 0.001, while for girls the mean difference was 2.7 mmHg with a p value of 0.009. For diastolic blood pressure the mean differences were 2.5 mmHg for the boys and 4.0 mmHg for girls with associated p values of 0.018 and 0.001, respectively.

Control for Confounding

The next stage of the analyses was to determine whether differences between the communities in other variables related to blood pressure might be confounding the results. Height and weight of the children, and educational level and occupation of parents were found to differ between the responding children of the two towns and to be related to systolic and/or diastolic blood pressure among boys and/or girls. Educational and occupational levels were combined into a single socioeconomic scale (SES) using the Hollingshead scoring system.

An analysis of covariance was used to determine whether the differential distribution of these confounding variables considered together could explain the differences in blood pressure found between the communities. The analysis of covariance controlled for height, weight, and social class. There were 32 children, 5.3 per cent, who could not be included in the analyses because of missing social class data, leaving an SES group of 576 pupils. Table 1 Section A compares the height, weight, social class adjusted mean differences for the group with SES data to the unadjusted mean differences for the same group and for the total sample. The adjustment for height, weight, and social class resulted in an increased mean difference for all four sex-blood pressure groups with the adjusted mean differences ranging from 3.2 to 4.5 mmHg.

Dietary Diaries

Codable 24-hour dietary diaries were collected from 312 (90.2 per cent) of the children in the high sodium community and 240 (91.6 per cent) of those in the low sodium community. Upon inspection, there were no obvious differences between the communities in the types or amounts of food eaten. However, Table 2 indicates the mean dietary intake of sodium, potassium, and calcium and shows the sodium/potassium ratio for the four sex/community groups. The sodium dietary intake is about 13 per cent higher and the potassium intake about 8 per cent higher in the high sodium community compared to the low sodium community for both sexes. Calcium intake is about 20 per cent higher for males and 16 per cent higher for females, whereas the sodium/potassium ratio is 5 per cent higher for the males and about 2-3 per cent higher for the females in the high sodium community. In absolute terms, the high sodium community pupils' intake per day is about 0.3 gm more sodium, 0.25 gm more potassium, and 0.2 gm more calcium.

The above figures do not include the sodium in the drinking water consumed either directly or when mixed with orange juice concentrate, coffee, soup, etc., or sodium absorbed by food from tap water used in cooking. Data on liquid consumption collected at a later time indicated that the third grade high sodium pupils consumed about 1 liter of tap water a day, obtaining approximately 120 mg of sodium a day from this source. Assuming a similar amount of water consumption in the low sodium community, the children there would receive 8 mg of sodium a day from their drinking water. However, it is important to note that when the sodium obtained from tap water is added to both communities' total dietary intake, then about 25 per cent of the difference in total sodium intake between communities is derived from this source: 24.4 per cent for males (112/459 mg) and 25.5 per cent for females (112/440 mg).

The sodium from tap water adds four per cent to the 2900 mg of dietary sodium in the high sodium community and .7 per cent to the 1100 mg of dietary sodium with low sodium community.

Table 1 Section B indicates that adjusting simultaneously for dietary intake of sodium, potassium, and calcium has little effect on differences in mean blood pressure between communities. On an individual level, the correlation between 24-hour dietary Na intake and systolic blood pressure was $r = .1929$ for boys and $r = -.0270$ for girls. For diastolic blood pressure, the correlations for boys and girls were $r = .0870$ and $r = -.0600$ respectively.

Most of the difference in dietary Na intake between communities was due to the school lunches served that day which contained 1049 mg of Na in the high sodium community and 816 mg of Na in the low sodium community, with 63 per cent and 60 per cent of the children respectively eating the school lunch. A further check of 151 school lunches in the past year indicated that the high Na community's school lunches averaged about 100 mg Na higher due in large part to a lack of freezer and cooler space and the consequent use of a large amount of canned rather than frozen or fresh vegetables.

First-Morning Urine Specimens

The originally drawn urine collection sub-sample of 200 students was found to be representative of the total sample in regard to blood pressure. Actual urine specimens were provided by 72 per cent of the boys and 70 per cent of the girls in the high sodium community and by 76 per cent of the boys and 62 per cent of the girls in the low sodium community. Although the response rates were similar in the two communities, the final sub-group for which urine specimens were obtained was not representative of the total sample in regard to mean blood pressure differences. Table 1 Section C clearly indicates this nonrepresentativeness with the increases in mean differences in the four cells of the specimen group ranging from 1.1 to 1.6 mmHg above the initial urine sub-

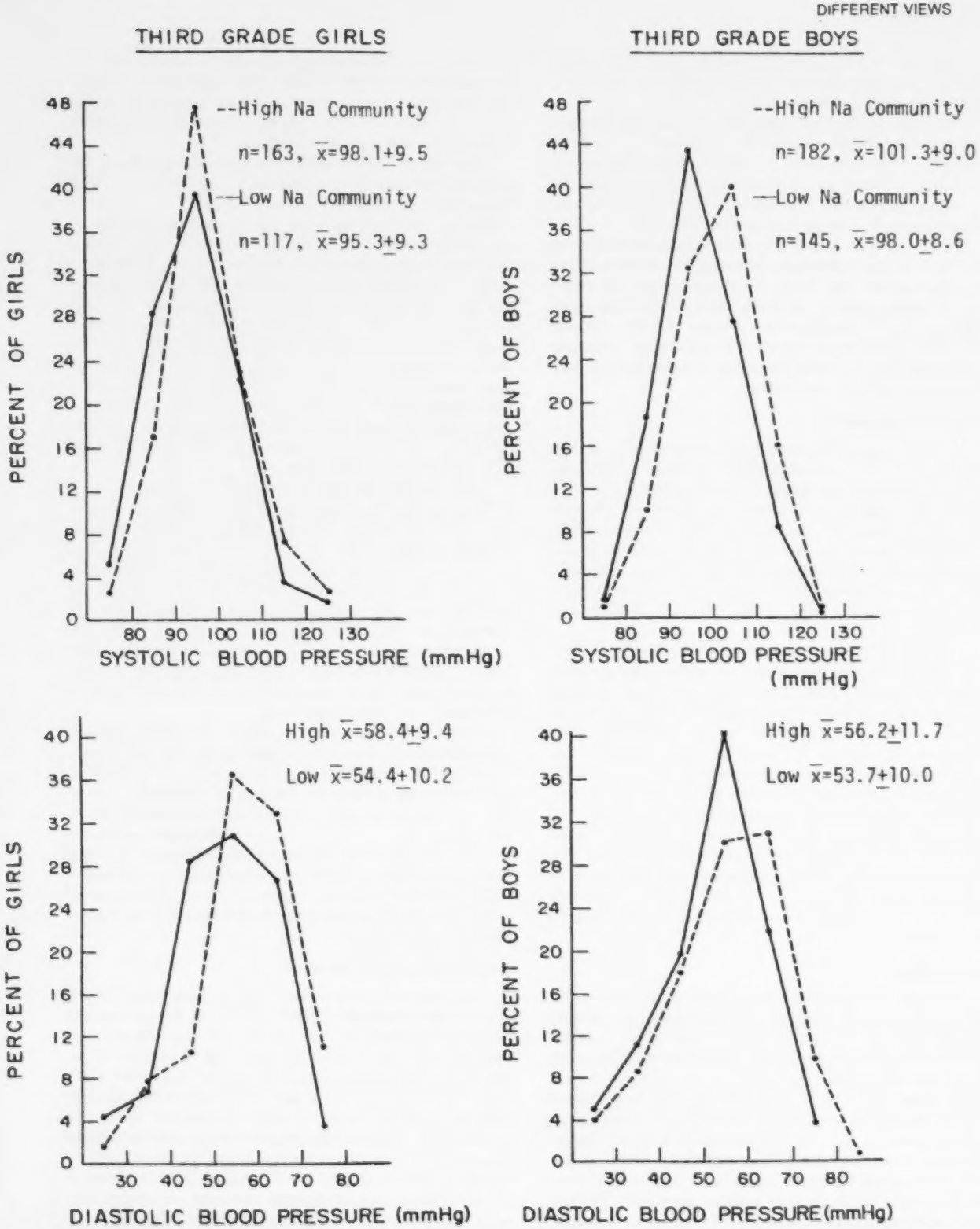


FIGURE 1—Percentage Distribution of Systolic and Diastolic Blood Pressure by Sex Group for a High Sodium Community and a Matched Low Sodium Community

TABLE 1—Differences in Mean Blood Pressure between the High and Low Sodium Communities for Various Sample Groups, both Unadjusted and Adjusted for Confounding Variables

Study Group	Condition	Sample Size	Male Systolic	Female Systolic	Male Diastolic	Female Diastolic	Range of p Values (1-Tail)
A							
Full Sample	Unadjusted	607	3.2	2.7	2.5	4.0	.018-.001
SES Group	Unadjusted	576	3.2	2.4	3.0	3.9	.020-.001
H-W-SC	Adjusted*	576	3.7	3.8	3.2	4.5	.007-.001
B							
Diet Sample	Unadjusted	527	3.7	2.4	3.8	4.0	.023-.001
H-W-SC	Adjusted*	527	4.4	3.8	4.1	4.5	.002-.001
H-W-SC-DNa DK-DCa	Adjusted†	527	4.1	3.9	4.0	4.6	.002-.001
C							
Urine Sample	Unadjusted	200	4.2	2.6	3.2	4.0	.077-.008
Specimen Grp.	Unadjusted	140	5.8	3.7	4.8	5.3	.050-.002
H-W-SC	Adjusted*	140	6.0	4.7	5.1	3.6	.055-.001
H-W-SC-UNa-Uk	Adjusted‡	140	6.2	5.1	5.7	4.4	.025-.001

*Differences were obtained by subtracting the mean average blood pressure (mm Hg) of the low sodium community from that of the high sodium community.
*Adjusted by analysis of covariance for height, weight, and social class.
†Adjusted by analysis of covariance for height, weight, social class, dietary sodium, potassium, and calcium intake.
‡Adjusted by analysis of covariance for height, weight, social class, urinary sodium, and potassium levels.

sample mean differences. This effect occurred because non-respondents in the high sodium community tended to be those with lower blood pressures while those non-respondents in the low sodium community tended to be those with high blood pressures. The result was an increase in the differences in mean blood pressure between the communities in the specimen group which destroyed the original representatives of the urine sample group.

The dietary intake for this urine specimen group showed a greater mean difference in sodium, potassium, calcium, and sodium/potassium ratio between the two communities when compared to the larger group for which dietary data were available. It should be noted also that the urine sample was not collected in concert with the dietary diary and the first-morning urine samples do not represent a 24-hour period.

TABLE 2—Difference in Dietary Intake of Sodium, Potassium, and the Sodium/Potassium Ratio between High and Low Sodium Communities

	Males						
	High Sodium Community (n = 160)		Low Sodium Community (n = 130)		Difference		p Value (2-Tail)
					Amount	%	
	$\bar{X}$	S.D.	$\bar{X}$	S.D.			
Sodium (mg)	2904	1088	2557	962	347	13.6	0.004
Potassium (mg)	3239	1190	2995	980	244	8.1	0.056
Calcium (mg)	1225	514	1023	379	202	19.7	0.001
Na/K Ratio	.958	.385	.914	.401	.044	4.8	0.366
	Females						
	(n = 152)		(n = 110)		Difference		p Value (2-Tail)
					Amount	%	
	$\bar{X}$	S.D.	$\bar{X}$	S.D.			
Sodium (mg)	2851	1135	2523	1013	328	13.0	0.015
Potassium (mg)	3036	986	2804	944	232	8.3	0.054
Calcium (mg)	1152	436	991	399	161	16.2	0.002
Na/K Ratio	1.000	.438	.976	.444	.024	2.5	0.610

The above caveats should be kept in mind in assessing differences between the two communities in urinary excretion of sodium, potassium, and in sodium/potassium ratios. Both males and females of the high sodium community showed a higher sodium excretion level (meq) than the low sodium community but with the difference for males three times that of the females. In contrast to the dietary diaries, potassium excretion was higher in the low sodium compared to the high sodium community. However, the sodium/potassium ratio differences were in the expected direction and highly statistically significantly different. When sodium and potassium excretion were controlled in an analysis of covariance, all four mean differences between communities increased slightly rather than decreasing as might be expected (Table 1 Section C). On an individual level, the correlation between urinary Na excretion and systolic blood pressure was r = .0282 for boys and r = .0880 for girls. The correlations of urinary Na excretion and diastolic blood pressure were r = .0043 and r = .1428 for boys and girls respectively.

Drinking Water Characteristics

Tap water samples from the homes of a sample of the third graders are being analyzed for 39 other water constituents in addition to sodium. An earlier analysis[42] examined the role of nine other elements in drinking water which had a potential for contributing to the differences in blood pressure between tenth grade students in the same two communities. In that study, the original association of high sodium levels with increased blood pressure levels was not explained when considering levels of barium, cadmium, chromium, copper, lead, lithium, vanadium, and zinc in the drinking water.

Discussion

An upshift in blood pressure found for tenth grade students was also found in a second study among third grade pupils in the same two communities, thereby demonstrating that the first study finding was not simply fortuitous. It is important to note that this higher mean blood pressure occurred in a population of children exhibiting a normally distributed blood pressure curve. The blood pressure increase appears to represent a general shift of the whole group to higher blood pressure levels rather than a small group of susceptibles at higher pressures exhibiting a response.

The 24-hour dietary diary data, available on over 90 per cent of the third grade pupils, showed a somewhat greater average dietary sodium and potassium intake as well as a larger sodium/potassium ratio in the high sodium community compared to the low sodium community. This difference on the community level is consistent with the aggregate correlations reported in the literature[23, 24] and is supportive of sodium as a factor in the difference in blood pressure between the communities. Since the difference in dietary sodium consumption was one-third of a gram between the communities, intake of one liter of high sodium tap water (120 mg/L) represented approximately 25 per cent of the *difference* in total sodium intake *between* the two communities even though the tap water added only four per cent to the dietary intake of Na in the high sodium community and 0.7 per cent Na to the dietary intake in the low sodium community.

On the individual level, the correlation between 24-hour dietary intake and blood pressure was quite low as again is commonly reported in the literature,[43-45] in contrast to the clear aggregate association reported above. There are a number of reasons why one would expect such a disparity between individual and group correlations. On the one hand, the measurement of individual dietary intake of sodium is fraught with problems: accuracy and completeness of reporting of food intake, wide daily individual variation in sodium intake not appropriately represented in a 24-hour sample of diet, and errors related to assigning standard (average) values for sodium to a class of food products. On the other hand, blood pressure recorded at a single seating (albeit three readings) is not necessarily a valid indication of usual blood pressure since there is considerable day-to-day individual variation. But most importantly, susceptibility to a salt-induced rise in blood pressure with age appears to be polygenetically distributed in the population. Until there is a way to measure the degree of individual genetic susceptibility to a salt-induced rise in blood pressure—and the range in such susceptibility is apparently large—direct correlations between sodium intake and blood pressure on the individual level must remain very modest. Recent work at Bowman Gray School of Medicine at Wake Forest University has demonstrated the existence of a low molecular weight peptide which inhibits renal ($Na^+ + K^+$) ATPase thus lending support to the hypothesis that renal sodium excretion is regulated by a humoral agent released or activated by extracellular fluid volume expansion.[46] This new "endoxin" may become a useful marker of genetic susceptibility to salt-induced elevations of blood pressure.

For the above reasons, the study reported here was designed to test the association between sodium intake and blood pressure at the community level. The differences found in blood pressure and sodium intake were based upon identical procedures used in both communities to obtain blood pressure and diet data. Although individuals might vary from day to day in blood pressure or sodium intake, the 300 pupils from each community would be expected to give a relatively stable picture of how the two groups of pupils differed from each other in these two factors.

An attempt to obtain first-morning urine samples from a representative sample of third graders was unsuccessful in that the 70 per cent who participated were differentially non-representative of the larger samples in the two communities in regard to blood pressure distribution. Any interpretation of the results was therefore of limited use.

Since blood pressure is a physiological parameter which may be affected by many variables, one must be cautious in attributing all effects to a single etiologic agent. However, our earlier tenth grade study and the current third grade study are certainly consistent in suggesting that sodium consumption in *drinking water and diet* contribute to the blood pressure differential between the two communities.

A major question still remaining is whether the relatively small differential in total dietary sodium intake of less than 0.5 gm per day, over at least several years, could have been

responsible for all the blood pressure differences found between the communities. The clinical literature is replete with studies indicating that large decreases (i.e., from 10 to 0.5 grams) in daily salt intake can reduce blood pressure in a matter of several weeks in hypertension-prone individuals.[27–31] But relatively little information has been gathered on the influence of chronic exposure to small differences in sodium intake in normotensive individuals.

A group of investigators in the Netherlands[47] recently (1979) obtained results supportive of those presented here (1977, 1978) when comparing the blood pressure of elementary school children in three communities differing in drinking water sodium levels. A long-term high Na community (160 mg/L) was compared to a long-term low Na community (30 mg/L) and to a short-term high Na community (changed from 23 mg/L to 150 mg/L a year before blood pressure was taken). Both high Na communities' children had statistically significantly higher mean blood pressures than the low Na community children, with the differences in systolic and diastolic blood pressure ranging from 1.8 to 4.0 mmHg after adjusting for confounding by weight, height, pulse rate, family history of hypertension, and time of day of blood pressure measurement. There were no dietary data collected, but 24-hour urinary Na excretion values for one day suggested a somewhat higher dietary intake of Na in the low Na drinking water community. From this study it would appear that short-term elevation in drinking water Na levels (of one year) may be sufficient for tap water Na to exert its influence on blood pressure.

Further work by the current authors continues to support the effect of sodium in tap water on blood pressure in children. Preliminary data from an experimental study* indicated that when high sodium tap water children were placed on low sodium bottled water there was a clear drop in blood pressure (for females at least) of several mmHg within the three month follow-up period in comparison to a group remaining on high sodium bottled water.

It may be that the sodium from local tap water makes a greater contribution to total dietary Na intake than is generally recognized. For instance, fresh or frozen vegetables with low levels of sodium when cooked in water with elevated levels of sodium absorb some of this sodium.[48] In addition, sodium may be more rapidly and efficiently absorbed via the gastrointestinal tract when imbibed in liquid form than when consumed in solid form. Such rapid absorption could put more stress on the homeostatic mechanisms controlling the extracellular fluid levels and other cardiovascular system adjustment mechanisms. Although not designed to test this hypothesis, a recent Japanese study of rats appears to indicate just such an enhanced effect of sodium imbibed in liquid compared to a similar amount taken in food.[17]

At the present time, the U.S. Environmental Protection Agency (EPA) lacks sufficient data on the effects of sodium levels in drinking water on blood pressure in normal populations. A currently proposed secondary drinking water standard would require public reporting of drinking water sodium levels which exceed the American Heart Association's limit of 20 mg/L recommended for ill persons on a restricted sodium diet.[49] The current work contributes to an increasing epidemiological data base assessing the health risks of chronic low level exposures to sodium in the drinking water. Such data on normal populations will be helpful in determining whether a primary drinking water standard for sodium is indeed necessary for protection for the general public.

*Paper presented by the authors at the Society of Epidemiological Research annual meeting, June 1980 in Minneapolis, MN.

REFERENCES

1. Sapirstein LA, Brandt WL, Drury DR: Production of hypertension in the rat by substituting hypertonic sodium chloride solution for drinking water. Proc Soc Exp Biol Med 1950; 73:82–85.
2. Toussaint C, Wolter R, Sibelle P: Effects de l'ingestion de grandes quantities de chlorure de sodium chez le rat. C R Soc Biol (Paris) 1953; 147:1637.
3. Meneely GR, Tucker RG, Darby WJ, *et al:* Chronic sodium chloride toxicity in the albino rat II. Occurrence of hypertension and of a syndrome of edema and renal failure. J Exptl Med 1953; 98:71–80.
4. Koletsky S: Hypertensive vascular disease produced by salt. Lab Invest 1958; 7:377–386.
5. Dahl LK: Effects of chronic excess salt feeding. J Exptl Med 1961; 114:231–236.
6. Fukuda TR: L'hypertension par le Sel Chez les Lapins et ses Relations Avec la Glande Surrenale. Union Med Can 1951; 80:1279–1283.
7. Lenel R, Katz LM, Rodbard S: Arterial hypertension in the chicken. Am J Physiol 1948; 152:557–562.
8. Seyle H: Production of nephrosclerosis in the fowl by sodium chloride. J Am Vet Med Assoc 1943; 103:140–143.
9. Langston JB, Guyton AC, Douglas BH, *et al:* Effects of changes in salt intake on arterial pressure and renal function in partially nephrectomized dogs 1963; 12:508–511.
10. Koletsky S: Pathogenesis of experimental hypertension induced by salt. Am J Cardiol 1961; 8:576–581.
11. Meneely GR, Ball COT, Youmans JB: Chronic sodium chloride toxicity: The protective effect of added potassium chloride. Ann Intern Med 1957; 47:263–273.
12. Meneely GR, Lemley-Stone J, Darby WJ: Changes in blood pressure and body sodium of rats fed sodium and potassium chloride. Am J Cardiol 1961; 8:527–532.
13. Dahl LK, Knudsen KD, Heine MA, *et al:* Effects of chronic excess salt ingestion. Modification of experimental hypertension in the rat by variations in the diet. Circ Res 1968; 22:11–18.
14. Haddy FJ, Overbeck HW: The role of humoral agents in volume expanded hypertension. Life Sci 1971; 19:935–947.
15. Bianchi G, Fox U, DiFrancesco GJ: Blood pressure changes produced by kidney cross-transplantation between spontaneously hypertensive rats (SHR) and normotensive rats (NR). Clin Sci Mol Med 1974; 47:435–438.
16. Dahl LK, Heine M, Thompson I: Genetic influence of the kidneys on blood pressure. Evidence from chronic renal homografts in rats with opposite predispositions to hypertension. Circ Res 1974; 40:94–101.
17. Aoki K, Yamori Y, Ooshima A: Effects of high or low sodium intake in spontaneously hypertensive rats. Jap Circl J 1972; 36:539–545.
18. Maddocks I: Blood pressures in Melanesians. Med J Aust 1967; 1:1123–1126.
19. Burns-Cox CJ, MacLean J: Splenomegaly and blood pressure in an Orange Asli community in West Malaysia. Am Heart J 1970; 80:718–720.
20. Cruz-Coke R, Etcheverry R, Nagel R: Influence on migration on blood pressure of Easter Islanders. Lancet 1964; 1:697–704.
21. Shaper AG: Cardiovascular disease in the tropics, III. Blood pressure and hypertension. Br Med J 1972; 3:805–807.
22. Kaminer B, Lutz WPW: Blood pressure in bushman of the Kalahari Desert. Circulation 1960; 22:289–295.

23. Page LB, Damon A, Moellering RC Jr: Antecedents of cardiovascular disease in six Solomon Islands societies. Circulation 1974; 49:1132–1146.
24. Prior IAM, Evans JG, Harvey HPB, *et al:* Sodium intake and blood pressure in two Polynesian populations. N Engl J Med 1968; 279:515–520.
25. Dahl LK: Salt and hypertension. Am J Clin Nutr 1972; 25:231–244.
26. Weinsier RL: Salt and the development of essential hypertension. Prev Med 1976; 5:7–14.
27. Kempner W: Treatment of hypertensive vascular disease with rice diet. Am J Med 1948; 4:545–577.
28. Dole VP, Dahl LK, Schwartz GC, *et al:* Dietary treatment of hypertension. Clinical and metabolic studies of patients on rice fruit diet. J Clin Invest 1950; 20:1189.
29. Dole VP, Dahl LK, Schwartz GC, *et al:* Dietary treatment of hypertension III. The effect of protein on appetite and weight. J Clin Invest 1953; 32:185.
30. Murphy RJF: The effect of rice diet on plasma volume and extracellular fluid space in hypertensive subjects. J Clin Invest 1950; 29:912–917.
31. Watkin DM, Froeb HF, Hatch FT, *et al:* Effects of diet in essential hypertension II. Results with unmodified Kempner rice diet in fifty hospitalized patients. Am J Med 1950; 9:441–493.
32. Ledingham JM: Distribution of water, sodium and potassium in heart and skeletal muscle in experimental renal hypertension in rats. Clin Sci 1953; 12:337.
33. Guyton AC, Coleman TG, Cowley AW, *et al:* A system analysis approach to understanding long-range hypertension. Circ Res 1974; 35:159.
34. Grollman A: The role of salt in health and disease. Amer J Cardiol 1961; 8:593–601.
35. Schroder HA: The role of salt in health and disease. Amer J Cardiol 1961; 8:593–601.
36. National Interim Primary Drinking Water Regulations. Federal Register Dec 24, 1975; 49(248):59576–59677.
37. American Heart Association: Your 500 milligram diet. New York: AHA, 1957.
38. Wolf HW, Esmond SE: Water quality for potable reuse of wastewater. Wtr Ser Works 1974; 121(2):48–52.
39. Fatula MI: Effect of water of high sodium chloride content on the incidence of arterial hypertension and temporary invalidity. Gig Sanit 1977; 2:7–11 (English translation).
40. Steinbach M, Constantinea M, Harnagea P: On the ecology of hypertension. Rev Roum Med 1974; 12(1):3–6.
41. Tuthill RW, Calabrese EJ: Elevated sodium levels in public drinking water as a contributor to elevated blood pressure levels in the community. Arch Environ Health 1979; 34:197–203.
42. Calabrese EJ, Tuthill RW: Elevated blood pressure levels and community drinking water characteristics. J Environ Sci Health 1978; A13(10):781.
43. Miall WE: Follow up study of arterial pressure in the population of a Welsh mining valley. Brit Med J 1958; 2:1204–1210.
44. Dawber TR, Kannel WB, Kagan A, *et al:* Environmental factors in hypertension. IN: The Epidemiology of Hypertension, pp 255–288, Stamler J, Stamler R, Pullman TM (eds): New York: Grune and Stratton, 1967.
45. Simpson FO, Waal-Manning HJ, Bolli P, *et al:* Relationship of blood pressure to sodium excretion in a population survey. Clinical Science and Molecular Medicine 1978; 55:373–375.
46. Gruber KA, Whitaker JM, Buckalew VM Jr: Endogenous digatilis-like substance in plasma of volume-expanded dogs. Nature 1980; 287:743–745.
47. Hofman, A, Vaandroger GF, Valkenburg HA: Increased blood pressure in school children related to high sodium levels in drinking water. Epidemiol and Community Health 1980; 34:179–181.
48. Rowan C: Effect of cooking water sodium levels upon the concentration of sodium and potassium in vegetables. Unpublished master's thesis. Amherst: University of Massachusetts, September 1980.
49. Federal Register. Proposed Regulations. July 19, 1970: 44:140.

ACKNOWLEDGMENTS

This research was supported by U.S. Environmental Protection Agency Grant #R-805612-02. The authors wish to acknowledge the contributions of the school administrators, teachers, parents and students whose generous cooperation made the study possible. The input of Mrs. Janelle Klar to the field operations and data preparation is also gratefully acknowledged.

TECHNICAL REPORT DATA
(Please read Instructions on the reverse before completin

1. REPORT NO.	2.	3. F
EPA-600/J-81-289	Journal Article	PB81-235772

4. TITLE AND SUBTITLE	5. REPORT DATE
Drinking Water Sodium and Blood Pressure in Children: A Second Look	July 1981
	6. PERFORMING ORGANIZATION CODE
7. AUTHOR(S)	8. PERFORMING ORGANIZATION REPORT NO.
R.W. Tuthill & E.J.Calabrese	
9. PERFORMING ORGANIZATION NAME AND ADDRESS	10. PROGRAM ELEMENT NO.
Division of Public Health School of Health Sciences Univ. of Massachusetts Amherst, MA 01003	11. CONTRACT/GRANT NO. R805612-02
12. SPONSORING AGENCY NAME AND ADDRESS	13. TYPE OF REPORT AND PERIOD COVERED
U.S. EPA Health Effects Research Laboratory 26 W. St. Clair St. Cincinnati, OH 45268	14. SPONSORING AGENCY CODE 600/10

15. SUPPLEMENTARY NOTES

Published in: AJPH 71(7):722-29(July 1981)

16. ABSTRACT

A previous study by the current authors demonstrated a statistically significant and clinically important elevation of 3-5 mmHg in mean systolic and diastolic blood pressure in high school sophomores in a community with 108 mg/L of sodium in the water supply when compared to their peers in an appropriately matched community with 8 mg/L of sodium. The current investigation, employing identical techniques but studying third graders in the same two communities, showed similar results. This second look considered dietary intake and urinary excretion of sodium. Since the difference in 24-hour dietary sodium consumption was 300 milligrams between the communities, an intake of one liter of high sodium tap water represented approximately 25 per cent of the difference in total sodium intake between the two communities. These studies suggest that sodium consumption in both drinking water and diet may be contributing to the difference in blood pressure distributions among the normotensive children in the two communities.

17. KEY WORDS AND DOCUMENT ANALYSIS

a. DESCRIPTORS	b. IDENTIFIERS/OPEN ENDED TERMS	c. COSATI Field/Group

REPRODUCED BY
NATIONAL TECHNICAL
INFORMATION SERVICE
U.S. DEPARTMENT OF COMMERCE
SPRINGFIELD, VA 22161

18. DISTRIBUTION STATEMENT	19. SECURITY CLASS *(This Report)*	21. NO. OF PAGES
Release to Public	Unclassified	
	20. SECURITY CLASS *(This page)*	22. PRICE
	Unclassified	